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Original Article

The impact of weight reduction in the prevention of the progression of obstructive sleep apnea: an explanatory analysis of a 5-year observational follow-up trial

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ABSTRACT

Background: Obstructive sleep apnea (OSA) is a chronic progressive disease, and it is well-documented that severe OSA is associated with an increased cardiovascular morbidity and mortality. Weight reduction has been shown to improve OSA; however, we need further evidence to determine if it may prevent the progression of OSA in the long term. The aim of our study was to assess the impact of weight change during a 5-year observational follow-up of an original 1-year randomized controlled trial.
Methods: The participants were divided into the two groups according to the weight change at 5-year follow-up using the 5% weight loss as a cutoff point, which was later referred to as the successful ($n = 20$) or unsuccessful groups ($n = 27$). The change in apnea–hypopnea index (AHI) was the main objective outcome variable.
Results: Fifty-seven patients participated in the 5-year follow-up. At 5 years from the baseline, the change in AHI between the groups was significant in the successful group (−3.5 [95% confidence interval {CI}, −6.1 to −0.9]) compared with the unsuccessful group (5.0 [95% CI, 2.0–8.5]) ($P = .002$). Successful weight reduction achieved an 80% reduction in the incidence of progression of OSA compared to the unsuccessful group (log-rank test, $P = .016$).
Conclusions: A moderate but sustained weight reduction can prevent the progression of the disease or even cure mild OSA in obese patients.

1. Introduction

Obesity has become an increasing health concern in recent decades. It is now well-known that obesity is associated with increased morbidity and mortality, in particular from cardiovascular and metabolic diseases [1,2]. Obesity also is the most important risk factor for obstructive sleep apnea (OSA); in fact, most OSA patients (at least 2 out of 3) are obese [3–5]. OSA is a chronic progressive disease and particularly the more severe stages of OSA have been linked to an increased risk for cardiovascular morbidity and mortality [6,7]. In the first randomized study conducted on the effects of weight loss on OSA, we demonstrated that a 1-year lifestyle intervention, which included an early weight reduction program, represented a feasible and effective treatment for overweight and obese participants with mild OSA [8]. These findings have been subsequently confirmed by two randomized studies, one conducted in obese OSA patients with type two diabetes mellitus (DM) and the

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other in patients with moderate to severe OSA using continuous positive airway pressure therapy [9,10].

Furthermore, our 2-year follow-up study demonstrated that the favorable changes achieved by a supervised lifestyle intervention during the intervention could be sustained for at least 1 year after the discontinuation of the active intervention [11]. In a recent randomized, 5-year, observational, postintervention follow-up study [12], we revealed that supervised lifestyle intervention based weight reduction (i.e., a healthy diet, increased physical activity) represented an effective treatment to prevent the progression of OSA when initiated in early phases of the disorder. These findings were recently supported by another recent long-term follow-up of 3 years [13]. Although these data were encouraging and weight loss is now recommended in all clinical guidelines on OSA, the efficacy of weight reduction as a treatment of OSA may still be underrated by many clinicians. Clinicians commonly believe that any weight loss could be temporary and would return after stopping the active lifestyle counseling; they also believe that this change could result in a re-exacerbation or worsening of OSA in most patients.

The main objective of our report was to extend the assessment of the postinterventional results conducted during the 5-year follow-up regarding the effect of weight loss and physical activity on OSA. To our knowledge, the effect of achieving the weight loss goal and sustaining it for years after the end of actual intervention has not been previously demonstrated. Percentage weight loss provides an easily measured goal for the intervention participants, and the number of participants who achieve a predetermined percentage of weight loss could offer a useful performance indicator for monitoring the efficacy of the intervention. Furthermore, the percentage also is an easily understood goal for the patients while aiming for weight loss. Thus weight loss $\geqslant$5% was the cutoff point we used in our study. We hypothesized that a successful and sustained weight reduction could prevent the progression of OSA.

2. Methods

Our paper is a detailed and extended secondary analysis of the original 5-year, controlled, randomized, follow-up trial examining the prevention of the progression of OSA [12]. Participants in the intervention group had received a 1-year lifestyle intervention including an initial weight reduction program with 12 weeks on a very low calorie diet. In the control group, only three general dietary and exercise counseling sessions were provided. The design of the study was previously reported in detail [8]. In our analysis on the association between weight change and OSA, the data on all participants were pooled and subdivided into two groups according to the body weight change at the 5-year follow-up using the 5% weight loss as a cutoff point, which was later referred to as successful or unsuccessful groups (Fig 1) [14,15]. The study protocol was approved by the Research Ethics Committee of the Hospital District of Northern Savo, Kuopio, Finland. The participants were given both oral and written information about the trial protocol and they provided a signed informed consent. The recruitment was planned for the previous 2 years and started in October 2004 and ended in December 2006.

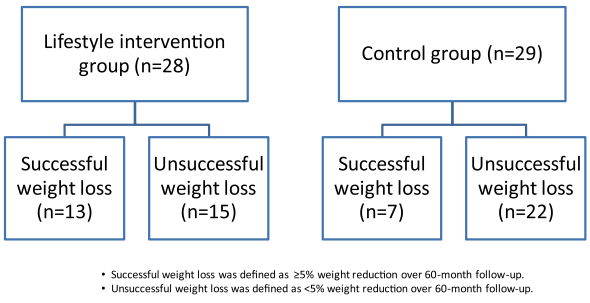

Fig. 1. Two groups according to the body weight change at 5-year follow-up using the 5% weight loss as the cutoff point (successful or unsuccessful group).

2.1. Participants

The study was conducted in Kuopio University Hospital, Finland. The study participants were consecutively recruited from the patients referred to the outpatient departments of Otorhinolaryngology and Respiratory Medicine of the Kuopio University Hospital due to a clinical suspicion of sleep-disordered breathing. They were assigned to undergo nocturnal cardiorespiratory monitoring. Weight and height were measured and the upper airways of the patients were inspected. The inclusion criteria for the initial trial were ages of 18–65 years, body mass index (BMI) of 26–40 kg/m^2, and apnea–hypopnea index (AHI) of 5–15 events per hour. The primary outcome measure was the magnitude of change in AHI during the follow-up period. The secondary outcome measures were changes in symptoms related to OSA and metabolic parameters. During the 5-year period, 24 patients dropped out of the study.

2.2. Intervention

In the original randomized study, the main goals of the dietary intervention were to reduce dietary fat to <30% of total energy and to increase the intake of fruits, vegetables, poultry, fish, and lean meat; they also needed to limit the consumption of dairy fats, fatty meats, and desserts. A detailed description of the implementation of the intervention has been previously reported [8]. In addition to the dietary counseling, the participants in the intervention group were recommended to increase their overall level of daily physical activity and endurance exercise, such as walking, skiing, jogging, or swimming. The frequency of physical activity of the participants was self-reported at the follow-up visits.

Based on the answers, the amount of physical activity was categorized to sufficient (i.e., 30 min or more exercise at least three times/week) or insufficient. The participants in the control group were only given standard care consisting of general verbal and written information about diet and physical activity at the baseline, 3-month, and 12-month follow-ups by the study nurse and physician without any specific individualized advice. During the next 4 years, no intervention or advice was offered to either of the randomization groups including at the 24-month follow-up. The study nurse regularly checked that the participants did not receive any co-intervention for OSA other than that specified in the study design.

2.3. Procedures and measurements

Nocturnal cardiorespiratory monitoring by Embletta® (Embla, Broomfield, CO) was conducted at the participants' homes in accordance with accepted guidelines for diagnosing OSA [16]. Recordings were manually evaluated and the two trained physicians (JKo, JRa) were blinded to clinical status and group. Apnea was defined as a cessation (more than 90%) of airflow for more than 10 s. Hypopnea was defined as a reduction (more than 30%) of airflow for more than 10 s with oxygen desaturation of $\geqslant$4%. AHI was defined as the number of apneas and hypopneas per hour; and mild OSA was defined as AHI of 5–15 events per hour, moderate as 15–30 events per hour, and severe as >30 events per hour [16]. The OSA was considered as objectively cured when AHI was <5 events per hour.

Validated questionnaires were used to screen for the intensity of snoring (Snore Outcomes Survey [SOS]) and daytime sleepiness (Epworth Sleepiness Scale [ESS]) [17,18]. The participants also

were asked if their bed partners noticed breathing pauses during the participant's sleep, and subsequently these were classified as witnessed apneas. Blood samples for biochemical assays were collected from participants after fasting for at least 12 h. All the biochemical measurements were performed in the Laboratory of Clinical Chemistry of Kuopio University Hospital. Serum cholesterol, high-density lipid (HDL) cholesterol, serum triglycerides, and plasma glucose were determined from fresh serum samples by using an automated analyzer system (Konelab 60 Analyzer, ThermoFisher Scientific, Waltham, MA), and serum insulin was measured with a fluoroimmunoassay system (Wallac, PerkinElmer, Waltham, MA).

2.4. Statistical analysis

The explanatory analyses of weight loss maintenance were performed after dividing the entire study population into patients with successful weight reduction (defined as weight reduction of $\geqslant$5% from baseline) and patients with unsuccessful weight reduction, irrespective of their original treatment group (intervention or control) (Fig. 1). The mean values and standard deviations were used to describe the baseline characteristics of the two groups. Fisher exact tests or *t* tests were used to assess equality between the treatment groups. Changes in sleep recordings and anthropometric measures during the follow-up were calculated by subtracting the baseline measurement from the 5-year measurement, and the mean difference in changes between the groups was calculated. The statistical significance of differences in changes between the groups was assessed with *t* tests, and additionally with analysis of covariance. In the analysis of covariance models baseline differences in age, sex, BMI, and the baseline level of the respective variables were considered by including these variables as explanatory variables into the model. Age and BMI were included in the models as continuous variables and sex as a dichotomous variable.

Kaplan–Meier survival curves were calculated to estimate the probability of the progression of mild OSA to moderate or severe OSA in the two groups. Participants who were lost during follow-up were treated as censored observations. No data were missing because of unsuccessful cardiorespiratory recordings. The difference between the survival curves was tested with the log-rank test. The Cox proportional hazard model was used to estimate the hazard ratio (HR) for the progression of OSA in those who achieved the weight loss goal of $\geqslant$5% at 12 months; those achieved this goal at 12 months and maintained it at 24 months; and those who achieved the goal at 12 months, maintained at 24 months, and again at 60 months with those who did not achieve these goals. The proportionality assumption of the model was assessed with graphical methods (i.e., the log–log plot). All comparisons between the treatment groups were based on the intention-to-treat principle, and all analyses were based on the 57 participants who attended the 5-year follow-up. In addition, separate analyses for the association of change in selected clinical variables to changes in AHI were assesses using logistic and linear regression, respectively. In the models, AHI was included as dependent variable, and the changes in BMI and of the concentrations of plasma glucose, serum insulin, HDL cholesterol, serum triglycerides, and serum alanine aminotransferase were included as predictors. Analyses were conducted with the statistics package Stata (StataCorp LP [2005], Stata Statistical Software: Release 9.2, College Station, TX).

3. Results

A total of 81 participants with OSA were originally randomized into our study. The baseline characteristics are shown in Tables 1 and 2. There were no differences in the baseline characteristics between successful and unsuccessful weight loss group (data not shown). Furthermore, there were no differences in the characteristics at baseline, 12-, or 24-month follow-up in individuals who participated in the entire 60-month follow-up compared to those who dropped out earlier (data not shown).

3.1. Follow-up data

3.1.1. Improvement in OSA according to weight reduction

The majority (*n* = 13/20; 65%) of the participants who were considered successful (weight loss $\geqslant$5% from baseline) in achieving

Table 1
Patient demographics at baseline. The data represent mean values with standard deviation (SD) or frequencies (%).

	Control group	Intervention group	*P* value
Study sample, randomized, *n*	41	40	
Complete 5-year follow-up, *n* (dropout %)	29 (29)	28 (30)	1.00
Gender, men/women, *n*	22/7	20/8	.77
Age, y	51.4 (8.1)	52.4 (8.6)	.66
Weight, kg	91.3 (11.6)	100.3 (13.6)	.01
BMI, kg/m^2	31.4 (3.1)	33.5 (3.3)	.02
Waist circumference, cm	105.7 (8.0)	112.0 (9.5)	.01
Plasma glucose, fasting, mmol/L	6.2 (1.8)	5.9 (0.9)	.46
Serum insulin, μIU/mL	10.8 (4.7)	13.7 (7.9)	.12
Serum cholesterol, mmol/L	4.6 (1.0)	4.7 (0.6)	.67
HDL cholesterol, mmol/L	1.1 (0.4)	1.0 (0.2)	.23
Serum triglycerides, mmol/L	1.6 (0.9)	1.7 (0.8)	.51
Serum alanine aminotransferase, U/L	33.6 (25.9)	38.0 (21.6)	.49
Systolic blood pressure, mmHg	130.7 (12.6)	130.1 (9.2)	1.00
Diastolic blood pressure, mmHg	81.4 (8.2)	82.0 (8.8)	.73
SOS, points	60.1 (14.8)	50.8 (12.3)	.01
ESS, points	9.9 (5.0)	10.1 (5.0)	.81
Witnessed apneas, *n* (%)	24[#] (89)	23[#] (100)	.24
Smoking, *n* (%)	5 (17)	5 (18)	1.00
Menopause, *n* (%)	7 (24)	8 (29)	.78
Substantial consumption of alcohol, *n* (%)	2 (7)	5 (18)	.42
Antihypertensive medication, *n* (%)	12 (41)	15 (54)	.43
Diabetes medication, *n* (%)	3 (10)	4 (14)	.71
Cholesterol medication, *n* (%)	14 (48)	6 (21)	.052

Abbreviations: y, years; BMI, body mass index (kg/m^2); SOS, Snore Outcomes Survey; ESS, Epworth Sleepiness Scale.
P value: Fisher exact test or *t* test for equivalence between groups.
[#] In witnessed apneas (*n* = 50), data missing from seven patients.

Table 2
Findings in the cardiorespiratory variables at baseline. The data represent mean values with standard deviation.

	Control group	Intervention group	*P* value
Study sample, randomized, *n*	41	40	
Complete 5-year follow-up	29	28	1.00
AHI, total	9.6 (2.9)	9.5 (3.0)	.96
Sleep recording time (min)	441.1	401.6	.12
AHI, supine	22.5 (16.0)	19.6 (15.2)	.52
Percentage of supine recording	31.1 (25.3)	39.8 (23.5)	.26
AHI, other positions than supine	5.1 (3.9)	6.9 (7.1)	.34
Mean SaO_2 (%)	94.3 (1.3)	94.0 (1.4)	.34
Time with mean SaO_2 below 90% (min)	6.7 (16.1)	9.7 (19.9)	.56
Percentage with SaO_2 below 90%	1.6 (3.6)	2.2 (5.1)	.61
Heart rate (beats/min)	60.3 (7.2)	59.4 (8.5)	.79

Abbreviations: AHI, apnea–hypopnea index (the number of apnea–hypopnea events per hour); min, minutes; SaO_2, arterial oxygen saturation.
P value: *t* test for equivalence between groups.

and sustaining weight reduction throughout the 5 years belonged to the lifestyle intervention group (Fig. 1). The mean AHI at the 5-year follow-up was 6.3 events per hour in the participants with successful weight reduction and 14.6 in those with unsuccessful weight reduction ($P = .001$). Furthermore, there was a statistically significant difference in the mean change of AHI during the follow-up between the successful and unsuccessful group (Table 3). The originally mild OSA was objectively cured in 50% ($n = 10/20$) of participants in the successful weight reduction group, compared to only 11% ($n = 4/37$) in those with unsuccessful weight reduction ($P = .003$).

Over the entire 5-year follow-up period, OSA had progressed from mild to moderate in only two participants in the successful weight reduction group but in 13 participants (and severe OSA in two participants) with unsuccessful weight reduction. Cox regression analysis showed that participants who achieved the $\geqslant$5% weight loss after 1 year had corresponding HR 0.40 (95% confidence interval [CI], 0.16–1.03; $P = .057$) and those who maintained their weight loss for 1 year (HR, 0.33 [95% CI, 0.12–0.92]; $P = .034$). For those participants who maintained the achieved weight loss at the 5-year follow-up, the incidence rates were 0.2 (95% CI, 0.0–0.6) and 0.7 (95% CI, 0.4–1.2) per 100 individuals-years in the original intervention and control groups, respectively (log-rank test, $P = .016$). The corresponding HR was 0.20 (95% CI, 0.05–0.87) (Fig. 2). There also were significant improvements in symptoms (i.e., SOS, ESS) related to OSA and in most of the other cardiorespiratory parameters favoring the successful group; however, there were no differences detected between the groups in statistical terms (Table 3). When weight loss and physical activity were simultaneously considered with regard to the association with reduction in AHI, only weight reduction was significant. Successful weight loss resulted in significant improvements in the concentrations of plasma insulin, HDL cholesterol, serum triglycerides, and serum alanine aminotransferase (Table 3).

To evaluate if the change in BMI or the changes in the metabolic variables explained the change in AHI, a multivariate regression model was used. With this model, 51.2% ($P < .0001$) of the variance of AHI could be explained. The change in BMI most adequately explained the change in AHI ($t = 3.867$; $P = .000$), followed by changes in plasma insulin ($t = 3.422$; $P = .001$), HDL cholesterol ($t = 2.986$;

Table 3
Changes in measurements from the baseline to the 60-month follow-up. The data represent mean changes with standard deviations or proportions when designated. Difference refers to the mean change between the successful weight reduction group (weight reduction $\geqslant$5% from the baseline) compared to the unsuccessful weight reduction group.

	Unsuccessful group	Successful group	Difference	*P* value	*P* value adjusted
Number of patients with follow-up data	37	20			
AHI, total	5.2 (9.7)	−3.5 (5.5)	−8.8	<.001	.002
AHI, supine	5.7 (29.9)	−1.7 (15.6)	−7.4	.35	.12
AHI, other position than supine	3.7 (6.3)	−3.5 (4.3)	−7.1	.009	.058
Percentage of supine recording	0.9 (28.5)	−1.1 (18.5)	−2.0	.81	.49
Mean SaO_2 (%)	−0.8 (1.7)	−0.3 (1.4)	0.5	.23	.22
Mean SaO_2 below 90%, (min)	26.8 (65.5)	−9.0 (26.6)	−35.8	.04	.11
Heart rate (beats/min)	2.9 (5.8)	−1.9 (5.0)	−4.8	.056	.062
Weight, kg	2.2 (6.2)	−10.9 (5.3)	−13.1	<.001	<.001
BMI, kg/m^2	0.7 (2.1)	−3.7 (1.7)	−3.4	<.001	<.001
Waist circumference, cm	2.5 (6.3)	−9.0 (6.4)	−11.5	<.001	<.001
Plasma glucose, mmol/L	0.04 (1.2)	−0.1 (0.4)	0.06	.58	.039
Plasma insulin, μIU/mL	5.2 (10.4)	−4.5 (7.6)	−9.7	.001	.001
Serum cholesterol, mmol/L	0.01 (1.2)	0.3 (0.7)	0.29	.34	.85
HDL cholesterol, mmol/L	0.04 (0.3)	0.2 (0.2)	−0.2	.007	.011
Serum triglycerides, mmol/L	0.1 (1.0)	−0.4 (0.6)	−0.5	.055	.011
Serum alanine aminotransferase, U/L	4.5 (26.6)	−10.7 (19.5)	−15.2	.03	.012
Systolic blood pressure, mmHg	0.7 (14.1)	0.3 (25.7)	−0.4	.96	.74
Diastolic blood pressure, mmHg	0.0 (9.0)	−3.3 (9.7)	−3.3	.45	.81
SOS, points	16.3 (25.2)	19.7 (20.2)	3.4	.61	.50
ESS, points	−2.6 (3.8)	−3.7 (4.3)	−1.1	.31	.56

Abbreviations: AHI, apnea–hypopnea index; SaO_2 = arterial oxygen saturation; BMI, body mass index; min, minutes; SOS, Snore Outcomes Survey; ESS, Epworth Sleepiness Scale.
P value: Fisher exact test or *t* test for equal change between groups.
P value adjusted: test for equal change between groups, adjusted for age, sex, BMI, and baseline level of the respective variable.

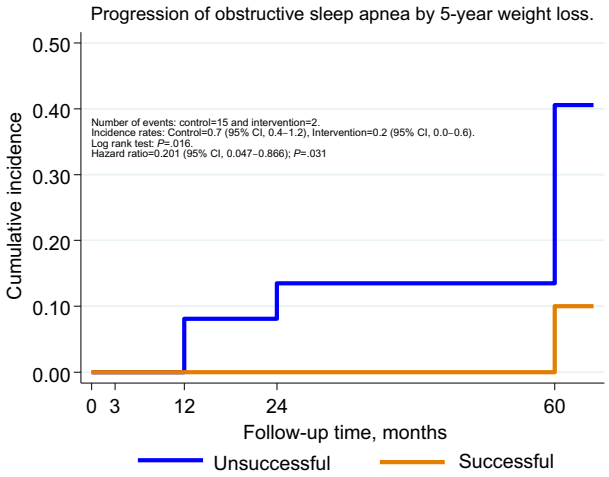

Fig. 2. Progression of obstructive sleep apnea (OSA) according to whether or not there was successful weight reduction during the postintervention follow-up period. The results are displayed as Kaplan–Meier estimates of probability for the progression of OSA. *Abbreviation:* CI, confidence interval.

$P = .005$), and plasma glucose ($t = 2.620$; $P = .012$) concentrations. Changes in serum triglyceride and serum alanine aminotransferase concentrations did not significantly enter the model.

3.1.2. Improvement in OSA according to original treatment group

Despite the original treatment group (i.e., either supervised lifestyle intervention group or control group), no difference was detected regarding the improvement in OSA when the weight loss was successful. Participants ($n = 13/20$) with successful weight loss originally belonging to the intervention group and those ($n = 7/20$) belonging to the control group had a mean AHI of 6.4 events per hour (−3.3 mean change from the baseline) and 6.9 events per hour (−3.9 mean change from the baseline), respectively. However, when the weight loss was considered unsuccessful, participants originally belonging to the intervention group had a mean AHI of 10.8 events per hour (1.1 mean change from the baseline) compared with participants belonging to the control group who had mean AHI of 17.0 events per hour (7.8 mean change from the baseline) ($P = .045$). Furthermore, nine participants (three with unsuccessful weight loss) from the intervention group (32%), and five participants (one with unsuccessful weight loss) from the control

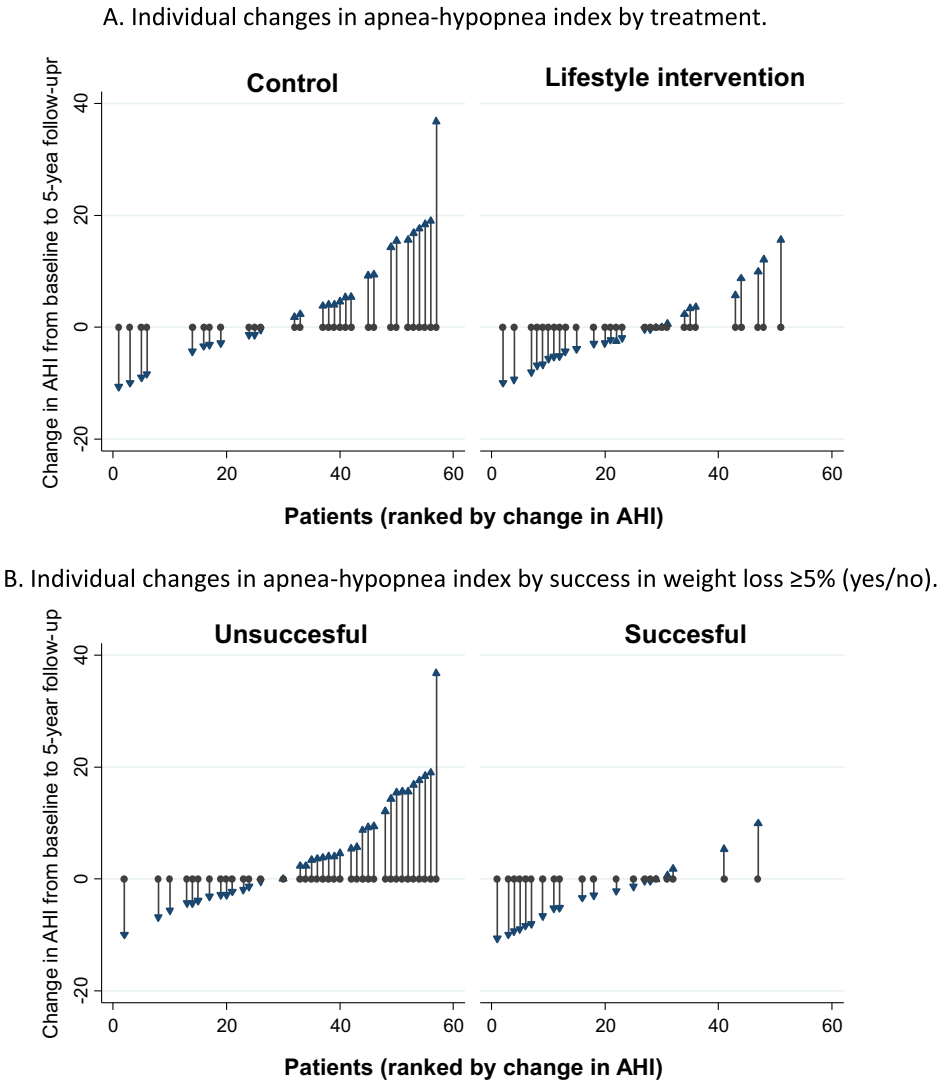

Fig. 3. Individual changes in apnea–hypopnea index (AHI) from baseline to 5-year follow-up in the lifestyle intervention vs control groups (A) and in the successful weight reduction vs unsuccessful weight reduction groups (B).

group (17%) were considered cured from OSA at the 5-year follow-up (Fig. 3).

3.1.3. Adverse events

No abnormalities in clinical variables requiring further action were observed during the 5-year follow-up period. The two participants with severe OSA at the 5-year follow-up were scheduled to undergo continuous positive airway pressure therapy.

4. Discussion

Our study provides long-term evidence that sustained weight reduction can result in significant long-term improvements of OSA in overweight patients and can prevent the progression of OSA. In the successful weight reduction group, a marked decrease in the AHI value was achieved from the baseline with every second participant being considered as objectively cured (i.e., they had AHI <5 events per hour). Furthermore, the disease progressed to moderate OSA over the 5-year follow-up in only two participants. In the unsuccessful weight reduction group, there was a significant increase (>50%) in the mean AHI, and the disease progressed in 15 of 37 participants. Overall, a successful weight loss reduced the risk for the progression of OSA by 80%. In addition to the improvement in AHI, significant improvements from baseline also were found in the symptoms related to OSA, as well as in the key cardiometabolic parameters during the follow-up period.

In clinical work, the association between OSA and metabolic disorders related to cardiometabolic syndromes needs to be considered. The interactions between OSA, type two DM, and metabolic syndrome are complex and multifactorial, but they clearly are influenced by one common feature in all of these disorders (i.e., obesity, particularly central obesity). Often these disturbances are present in the same individual and it has been postulated that the coexistence of these conditions could have an even more devastating impact on the serious cardiometabolic consequences than any one of these conditions on their own [12]. Our findings demonstrate that a sustained weight loss resulted in a major improvement not only in respiratory function but also in metabolic disorders in overweight participants with OSA, suggesting an improvement in insulin resistance. Interestingly these changes in the concentrations of plasma glucose, plasma insulin, and HDL cholesterol also explained the improvement in AHI beyond the benefit achieved by weight reduction. This novel finding suggests that not only weight reduction but also its metabolic consequences seemed to have beneficial effects on AHI. This finding is further supportive of the proposal that weight reduction and increased physical activity should be offered as the first-line treatment to obese OSA patients with metabolic disorders [12,13].

OSA tends to worsen with time, and therefore individuals with mild OSA may be considered to be at high risk for progression of the disease. One previous observational study noted that originally mild OSA in participants of the same baseline age, weight, AHI, and follow-up as in our present study deteriorated from a mean AHI of 9.1 events per hour (mild) to 21.7 events per hour (moderate), which was mostly due to weight gain [19]. Our results highlight that even moderate, but sustained weight reduction does matter. During the 5-year follow-up period, there was an 80% risk reduction in the progression of the OSA in participants who achieved a weight loss of $\geqslant$5%. In our study this weight loss is equivalent to 5 kg or more compared with those not achieving even this modest goal. Furthermore, the originally mild OSA had been objectively cured in 50% as compared with only one out of every 10 in those with unsuccessful weight reduction in the successful weight loss group. Based on current knowledge about the evolution of OSA, we believe that weight gain represents a high risk for further progression towards more severe disease, particularly in those patients with mild to moderate OSA who already have a partial obstruction of their upper airways due to anatomical or functional causes [19,20]. This finding is of importance, as it is well-documented that the severity of OSA is associated with an increased risk for cardiovascular morbidity and mortality [6,7].

A similar approach to that used in DM prevention studies with weight reduction programs also could be relevant to individuals with OSA with mild symptoms to interrupt disease progression, and thus to prevent the associated increased morbidity and mortality. Furthermore, it is said that it is more difficult to improve OSA by weight reduction than to further exacerbate OSA by weight gain [5,21]. This finding highlights the importance of maintaining normal body weight by providing relevant information to the public through prevention programs, motivating them to achieve early control of their condition and more active treatment of their obesity by changing lifestyles in cases of overweight individuals.

It has been suggested that changes in weight may have different modulatory effects on various breathing parameters, mainly on lateral AHI, and therefore on positional dominance [22]. Our results also support these findings, as the lateral AHI was found to improve, mostly related to the successful weight loss. Our study also supports the earlier well-characterized findings on the association between regular physical activity and reduced likelihood of sleep-disordered breathing [23]. The lifestyle intervention group achieved a successful weight reduction more often, and overall the participants achieving the weight loss goal reported that they exercised more regularly.

Some limitations of our study must be addressed. Because the lifestyle intervention included both dietary and healthy lifestyle counseling and increased physical activity and the sample size was relatively small, it is difficult to differentiate between the impacts of the different components of the lifestyle changes. The study was conducted in individuals with mild OSA; therefore, our findings may not be directly generalizable to all OSA patients. Instead of using in-laboratory polysomnography, portable recording devices were used in our study. However, clinical guidelines for the use of portable monitors have been introduced and there is now a recommendation that portable monitoring may be used as an alternative to polysomnography for the diagnosis and treatment follow-up of OSA [24]. Therefore, these encouraging results need to be replicated in a larger randomized intervention study.

When the disease is still at an early stage, it is most likely that the organ systems have the capacity to recover from the adverse impact of OSA related to excessive body weight or the inexorable progression of the disease may at least be prevented. In addition to relieving the symptoms of OSA, weight reduction also improves many other obesity-related components of the cardiometabolic syndrome and helps in prevention of type two DM [14,15,25]. Our results demonstrate that sustained weight reduction is an effective treatment modality for obese people with mild OSA.

Contributors HT, JSE, MUU, and HGY were responsible for the general conception and study design. HT drafted the manuscript, with input from JSE, MPE, HGY, JTU and MUU. HT coordinated the study, MPE performed the statistical analyses. JSE, GSM, MPU, EVA, JKO, JRA, HTU, JSA, and TMA contributed to the clinical work related to the study and participated in interpreting the results and in editing of the manuscript. All authors have approved the current version.

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Conflict of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: http://dx.doi.org/10.1016/j.sleep.2013.11.786.

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